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Heart Failure and Cardiomyopathies

DONOR AND RECIPIENT RENAL DYSFUNCTION AND POST CARDIAC TRANSPLANT GRAFT SURVIVAL: INSIGHTS INTO RENO-CARDIAC INTERACTIONS

Poster Contributions
Hall C
Sunday, March 30, 2014, 3:45 p.m.-4:30 p.m.

Session Title: Approaches to Advanced Heart Failure: From VAD, Transplant, Palliative Care to New Perctutaneous Therapies
Abstract Category: 12. Heart Failure and Cardiomyopathies: Clinical
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Background: The major mode of death in patients with renal dysfunction (RD) is cardiovascular disease (CVD). Notably, there may be a causal effect of RD given that myocardial necrosis/apoptosis has been seen in animal models of RD. However, RD is also a marker of overall CVD severity. Cardiac transplantation provides an opportunity to study this as hearts are being transplanted in and out of the environment of RD: If irreversible myocardial damage occurs immediately with RD, as seen in animal models of acute kidney injury, transplantation of a heart from a donor with RD should yield reduced graft survival. However, if cardiac damage from RD develops gradually, transplantation of a healthy RD-free donor heart into a recipient with RD should yield an initial low risk period followed by high event rates months to years later.

Methods: Adult cardiac allograft recipients in the UNOS registry were studied (n=35,914). RD was defined as estimated glomerular filtration rate (eGFR) < 60 ml/min/1.73m2.

Results: RD was present in 17.2 % of donors and 39.4% of recipients with an overall worsening in eGFR over time in recipients ($p<0.001$). Donor characteristics known to cause or reflect myocardial damage such as ischemic time > 4 hours (adjusted HR 1.2, $p<0.001$), age > 50 years (adjusted HR=1.3, $p<0.001$), or ejection fraction ≤ 45% (adjusted HR 1.2, $p=0.03$) were associated with reduced graft survival. To the contrary, the risk associated with RD did not follow the heart as transplantation from a donor with RD did not reduce graft survival (adjusted HR=0.98, $p=0.44$). RD-free donor hearts placed into a recipient with RD paradoxically had the highest risk of graft dysfunction in the first 30 post-operative days (Adjusted HR 1.6, $p<0.001$). Subsequently, the hazard attributable to recipient RD (adjusted HR 1.2, $p<0.001$) did not increase over time ($p=0.8$) as would be expected with slow accumulation of myocardial damage from RD.

Conclusion: Transplantation of a heart in and out of the environment of RD was not associated with worsened outcomes in a manner consistent with a clinically meaningful direct effect of RD on the myocardium. These data provide additional support that RD primarily serves as a marker rather than a direct cause of CVD.